



Research report

Pure word deafness with auditory object agnosia after bilateral lesion of the superior temporal sulcus



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ABSTRACT

Based on results from functional imaging, cortex along the superior temporal sulcus (STS) has been suggested to subservise phoneme and pre-lexical speech perception. For vowel classification, both superior temporal plane (STP) and STS areas have been suggested relevant. Lesion of bilateral STS may conversely be expected to cause pure word deafness and possibly also impaired vowel classification. Here we studied a patient with bilateral STS lesions caused by ischemic strokes and relatively intact medial STPs to characterize the behavioral consequences of STS loss. The patient showed severe deficits in auditory speech perception, whereas his speech production was fluent and communication by written speech was grossly intact. Auditory-evoked fields in the STP were within normal limits on both sides, suggesting that major parts of the auditory cortex were functionally intact. Further studies showed that the patient had normal hearing thresholds and only mild disability in tests for telencephalic hearing disorder. Prominent deficits were discovered in an auditory-object classification task, where the patient performed four standard deviations below the control group. In marked contrast, performance in a vowel-classification task was intact. Auditory evoked fields showed enhanced responses for vowels compared to matched non-vowels within normal limits. Our results are consistent with the notion that cortex along STS is important for auditory speech perception, although it does not appear to be entirely speech specific. Formant analysis and single vowel classification, however, appear to be already implemented in auditory cortex on the STP.

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Abbreviations: AAT, Aachner Aphasia Test; ITD, interaural time difference; MEG, magnetoencephalography; MTG, middle temporal gyrus; PPDT, psychoacoustic pattern discrimination test; STG, superior temporal gyrus; STP, superior temporal plane; STS, superior temporal sulcus.

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1. Introduction

Central hearing disorders subsequent to auditory cortex lesions are often subtle and considered of minor significance for clinical neurology and neurorehabilitation. While hearing deficits are reliably found in patients with unilateral lesions of the auditory cortex when dichotic tests are applied (Biedermann, Bungert, Dorrscheidt, von Cramon, & Rubsamen, 2008; Blaettner, Scherg, & von Cramon, 1989; Gutschalk, Brandt, Bartsch, & Jansen, 2012; Musiek, 1983), these patients typically have unimpaired pure tone thresholds in both ears and intact speech perception unless other areas are additionally involved. Disability of auditory speech perception is frequently observed as part of the aphasia syndromes, in particular in Wernicke's aphasia (Robson, Grube, Lambon Ralph, Griffiths, & Sage, 2013). However, aphasia is never limited to disturbance of auditory speech perception, but also involves disability of speech production and understanding of written language (Alexander & Hillis, 2008). A more isolated disorder of speech perception is observed rather rarely and is known as “pure word deafness” (Kusmaul, 1877). While pure word deafness is relatively rare, it is one of the most disabling cortical hearing disorders. Hallmarks that dissociate pure word deafness from aphasia are relatively intact expressive language function, including spoken as well as written language, and the ability to understand written language.

Conversely, pure word deafness is often not limited to speech perception, but additionally involves some degree of auditory object agnosia or acquired amusia (Buchman, Garron, Trost-Cardamone, Wichter, & Schwartz, 1986), with few exceptions (Yaqub, Gascon, Al-Nosha, & Whitaker, 1988). While a number of case reports demonstrated auditory agnosia for environmental objects with relative intact auditory speech perception (Albert, Sparks, Von Stockert, & Sax, 1972; Saygin, Leech, & Dick, 2010), an anatomical separation between pure word deafness – or auditory verbal agnosia – and auditory object agnosia has never been established (Robert Slevc & Shell, 2015).

It has therefore been suggested to use only “word deafness” (Buchman et al., 1986), because ‘pure’ could be misunderstood to exclude other auditory deficits and agnosia rather than indicate lack of aphasia. Here, we continue to use pure word deafness to characterize a spectrum of disorders that necessarily include disability of auditory speech perception in the absence of other signs of aphasia, but which may typically include auditory object agnosia and some lower level auditory deficits (Auerbach, Allard, Naeser, Alexander, & Albert, 1982; Kusmaul, 1877). However, we demand that impaired speech perception cannot be attributed to elevation of sound-detection thresholds, on the other hand (Auerbach et al., 1982; Buchman et al., 1986), and that auditory awareness for the presence of sound sources remains intact.

This is in contrast to cortical deafness (Bahls, Chatrian, Meshner, Sumi, & Ruff, 1988; Özdamar, Kraus, & Curry, 1982), where awareness of sound presence is disturbed by cortical lesion. In analogy to cortical blindness, which is thought to be caused by complete lesion of V1 (Aldrich, Alessi, Beck, & Gilman, 1987; Celesia, Bushnell, Toleikis, & Brigell, 1991), it

is often inferred that cortical deafness would occur with bilateral lesion of the primary auditory cortex. However, primate (including human) auditory cortex comprises three functionally distinct primary-like fields (Morel, Garraghty, & Kaas, 1993; Morosan et al., 2001). These primary or core fields are surrounded by multiple belt and parabelt fields, which also receive projections from medial geniculate body, although in a different blending (Hackett, 2015). While patients with bilateral lesions that include putative primary auditory cortex in the medial 2/3 of Heschl's gyrus may initially fulfill the criteria for cortical deafness, they often regain awareness of sound presence and relatively normal pure tone thresholds, but show persistent deficits in basic frequency and intensity discrimination thresholds (Dykstra, Koh, Braida, & Tramo, 2012; Mendez & Geehan, 1988; Tramo, Shah, & Braida, 2002). Recovery from cortical deafness has also been observed in monkey subsequent to bilateral ablation of the auditory core and belt cortex (Heffner & Heffner, 1990), but with a persisting threshold elevation in the range of 30–44 dB. It therefore appears that persistent cortical deafness requires lesion of all cortex areas that receive projections from the medial geniculate body, including core, belt, and possibly even parabelt areas (Cavinato, Rigon, Volpato, Semenza, & Piccione, 2012; Engelien et al., 2000), the exact topography of which have not yet been settled in human (Hackett, 2015). A role of non-primary auditory cortex for auditory perceptual awareness is also supported by evoked response studies (Gutschalk, Micheyly, & Oxenham, 2008).

For patients with lesions of the auditory cortex but intact awareness of sound presence, the labels “cortical auditory disorder” (Kanshepolksy, Kelley, & Waggener, 1973; Mendez & Geehan, 1988) or “cortical deafness” (Michel, Peronnet, & Schott, 1980) have been suggested, whereby the latter seems problematic for the reasons outlined above. Note, that the clinical presentation of these patients is very similar to pure word deafness, and that an unambiguous separation of these entities has not yet been established.

Pure word deafness has also been associated with bilateral, but less extensive temporal lesions (Barrett, 1910; Buchman et al., 1986; Geschwind, 1965; Kanshepolksy et al., 1973; Wohlfart, Lindgren, & Jernelius, 1952; Yaqub et al., 1988). An early model of pure word deafness assumed a disconnection between Wernicke's area (Wernicke, 1874), thought to be the center of “auditory word representation”, and the input from auditory cortex (Lichtheim, 1885). This hypothesis was later refined as disconnection between bilateral auditory core and belt cortex (roughly corresponding to Brodmann areas 41 and 42) and Brodmann area 22 in posterior superior (and middle) temporal gyrus (STG and MTG) on the left (Geschwind, 1965). Based on the assumption that there is no strong transcallosal connection between left and right area 22, anatomical possibilities considered were subcortical destruction of the auditory transcallosal fibres combined with lesion of the auditory radiation on the left, or with lesions between areas 42 and 22 in the anterior STG. It was further argued that pure word deafness was so rare, in particular with unilateral lesions, because most lesions fulfilling these criteria also involved area 22 and therefore caused Wernicke's aphasia.

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